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Gastritis

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INDEX

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I.

UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
CALENDAR OF EVENTS
March 12 - 17, 1945

No. 62

Monday, March 12

9:00 - 10:00 Roentgenology-Medicine Conference; L. G. Rigler, C. J. Watson and Staff; Todd Amphitheater, U. H.

9:00 - 11:00 Obstetrics and Gynecology Conference; J. L. McKelvey and Staff; Interns Quarters, U. H.

12:30 - 1:30 Pathology Seminar; Acute Pancreatitis, D. W. Molander, 104 I. A.

Tuesday, March 13

9:00 - 10:00 Roentgenology-Pediatrics Conference; L. G. Rigler, I. McQuarrie and Staff; Eustis Amphitheater, U. H.

11:00 - 12:00 Urology Conference; C. D. Creevy and Staff; Main 515 U. H.

12:30 - 1:30 Pathology Conference; Autopsies; Pathology Staff; 104 I. A.

12:30 - 1:30 Physiology-Pharmacology Seminar; The Problem of Standards and Variability in Human Biology; Howard Alexander and Lester Erickson, 214 M. H.

4:00 - 5:00 Physiological Pathology of Surgical Diseases; Physiology and Surgery Staffs; Todd Amphitheater, U. H.

4:30 - 5:30 Obstetrics and Gynecology Conference; J. L. McKelvey and Staff; Station 54, U. H.

4:00 - 5:00 Pediatrics Grand Rounds; I. McQuarrie and Staff; W-205 U. H.

4:30 - 5:30 Ophthalmology Ward Rounds; Erling Hansen and Staff; E-534, U. H.

5:00 - 6:00 Roentgen Diagnosis Conference; K. W. Stenstrom and Leslie P. Anderson, 515 U. H.

Wednesday, March 14

9:00 - 11:00 Neuropsychiatry Seminar; J. C. McKinley and Staff; Station 60 Lounge, U. H.

11:00 - 12:00 Pathology-Medicine-Surgery Conference; Myocardial Infarction, Cushings Syndrome; E. T. Bell, C. J. Watson, O. H. Wangensteen and Staff; Todd Amphitheater, U. H.

12:30 - 1:30 Pediatrics Seminar; Congenital Megacolon; Dr. Lundgren; W-205 U.H.

12:30 - 5:30 Physiological Chemistry Literature Review; Staff; 116 M. H.

4:30 - 5:30 Neurophysiology Seminar; The Results of Physiological Neuronography; J. P. Murphy, 214 M. H.

Thursday, March 15

9:00 - 10:00 Medicine Case Presentation; C. J. Watson and Staff; Todd Amphitheater.

4:00 - 5:00 Pediatric Journal Club; Review of Current Literature; Staff; W-205 U. H.

4:30 - 5:30 Ophthalmology Ward Rounds; Erling Hansen and Staff; E-534, U. H.

5:00 - 6:00 Roentgenology Seminar; Results of the X-ray Therapy in Pituitary Tumors; Harry Mixer, M-515 U. H.

Friday, March 16

9:00 - 10:00 Medicine Grand Rounds; C. J. Watson and Staff; Todd Amphitheater, U.H.

10:00 - 12:00 Medicine Ward Rounds; C. J. Watson and Staff; E-214 U. H.

10:30 - 12:30 Otolaryngology Case Studies; L. R. Boies and Staff; Out-Patient Otolaryngology Department, U. H.

11:45 - 1:15 University of Minnesota Hospitals General Staff Meeting; External Eye Lesions; Erling S. Hansen; Powell Hall Recreation Room.

1:00 - 2:30 Dermatology and Syphilology; Presentation of Selected Cases of the Week; Henry E. Michelson and Staff; W-206, U. H.

1:30 - 3:00 Roentgenology-Neurosurgery Conference; H. O. Peterson, W. T. Peyton and Staff; Todd Amphitheater.

Saturday, March 17

8:00 - 9:00 Surgery Journal Club, O. H. Wangensteen and Staff; M-515 U. H.

9:00 - 10:00 Pediatrics Grand Rounds; I. McQuarrie and Staff, Eustis Amphitheater, U. H.

9:15 - 10:30 Surgery Roentgenology Conference; O. H. Wangensteen, L. G. Rigler and Staff; Todd Amphitheater, U. H.

9:00 - 10:00 Medicine Case Presentation; C. J. Watson and Staff; M-515 U. H.

10:00 - 12:00 Medicine Ward Rounds; C. J. Watson and Staff; E-221 U. H.

II. GASTRITIS

R. S. Ylvisaker

It will soon become apparent that the title to this presentation is somewhat of a misnomer. In the first place, it is impossible to cover a field which has become so complex and concerning which so much controversy has arisen. In the second place, no attempt will be made to discuss acute gastritis except to state that Faber and others have demonstrated beautifully that acute gastritis frequently accompanies acute infectious diseases and often results from local mechanical and chemical irritants and that it frequently leads to interference with gastric secretion and achlorhydria. In the third place, only a few of the highlights in the field of chronic gastritis will be mentioned and some of our experiences in the gastroscopic clinic at the University of Minnesota Hospitals will be brought out.

Probably the earliest anatomic description of gastritis was that by Broussais in 1823. He found pathologic changes to be present in almost all stomachs and small intestines at postmortem examination and it was not until Robert Carswell showed that most of these changes were due to postmortem digestion of the mucosa, that the diagnosis of chronic gastritis lost its great popularity.

Definite proof that chronic gastritis was an entity began to appear toward the end of the 19th century. Fenwick first described atrophy of the gastric mucosa in cases of pernicious anemia in 1877. Hayem in 1892 introduced bichromate solution into the stomach through a tube soon after death and was thereby able to study in some detail the histopathology of chronic gastritis.[1]

It remained, however, for Faber[2] and some of this co-workers and contemporaries to develop a method of fixation whereby the stomach mucosa could be studied in detail. They injected 10 per cent formalin directly through the abdominal wall into the viscera immediately after death and obtained immediate fixation. In order to determine what the normal stomach should look like they investigated altogether thirty formalin-fixed stomachs of newborn infants. These showed no interstitial cell infiltration. Faber goes on to state that the same was found in many older children and adults. When the stomach is normal only a fine network of fibrillary threads and a few scattered plasma cells or lymphocytes, but no true leucocytes, are found between the closely packed glands of the mucous membrane. A few lymphoid follicles are also seen but by no means always. This normal appearance is quite common in the corpus and fornix, but it is quite rare not to find gastritic spots especially in the pyloric area.

Faber then goes on to describe the changes that occur in acute and chronic gastritis but calls any sharp distinction between the two forced and artificial and reminds us of the overlapping which is seen between the two. Chronic gastritis will necessarily consist of acute and chronic processes; the acute - hyperemia and edema, leucocytic infiltration of the mucous membrane, degeneration of the glandular epithelium and erosions of the mucous membrane; the chronic - more the character of consequences of the degeneration and atrophy, and of the regenerative processes which the organism has been able to put into action.

He divides chronic gastritis into two distinct forms, the chronic erosive gastritis especially localized to the antrum, and the diffuse pangastritis which tends toward atrophy, the "gastritis progressiva atrophicans" of Lubarsch. The chronic erosive gastritis Faber describes as showing the usual macroscopic signs of inflammation, hyperemia, edema, and thickening of the folds, but the most striking feature being the more or less numerous erosions in various acute, chronic and healed stages. These latter, he states, are caused by polynuclear leucocytes invading the destroying the surface epithelium in patches, after which leucocytes and pus corpuscles are shed into the cavity of the stomach leaving a defect in the epithelium. Microscopically in addition to the erosions, the mucous membrane shows the usual signs of inflammation in these forms of gastritis - enor-

nous round-celled infiltration and numerous lymphoid follicles. In the more acute processes the infiltrating cells are largely polynuclear leucocytes and extravasations of blood into the tissues are often seen. The more chronic processes exhibit an increasing number of plasma cells and lymphocytes and the formation of fibrillary connective tissue surrounding the chronic erosions. The erosions usually do not penetrate the mucous membrane, but occasionally the muscularis mucosae and submucosa is penetrated and a small, true peptic ulcer is formed. Sooner or later, as the process goes on, the chronic changes predominate and eventually lead to atrophy.

The chronic, diffuse pangastritis is usually seen in cases of chronic anacidity. Macroscopically the mucous membrane is smoothed out and the folds small. The color is pale, and hyperemia having disappeared. Usually we find none of the chronic erosions. Microscopically, plasma cells and lymphocytes predominate in the cellular infiltration. As the chronic inflammation develops, atrophic processes occur and the gland epithelium degenerates and disappears, at first in isolated patches, later over extensive areas. The interstitial tissue takes on the character of granulation tissue which surrounds the gastric crypts and the thickness of the mucous membrane gradually diminishes sometimes to a fraction of its normal depth.

In some cases cysts develop and these may become numerous, leading to the descriptive diagnosis of chronic cystic gastritis. In others, the lymph follicles may be greatly increased in number suggesting a chronic follicular gastritis.

Regenerative processes also occur along with the degenerative, resulting in various changes. There may be proliferation of the surface epithelium forming fine villous prominences or there may be true hypertrophy into wart-like elevations which may be single or multiple in which case the condition may be termed gastritis polyposa. The specific gland cells of the corpus glands may be replaced by undifferentiated pseudo-pyloric gland cells. Also in most cases of chronic atrophic gastritis there occurs a more or less extensive and very characteristic transofrmation of the surface epithelium into a goblet cell epithelium resembling that found in the small intestine. Along with this metaplasia of the epithelium, the original glands may be replaced by typical Liberkühn glands which may even contain characteristic Paneth cells.

The original descriptions of Faber are here reviewed in some detail because I believe it is generally agreed that they form the basis of our present-day conception of the pathology of gastritis. Faber's work received ample confirmation by Konketzny, Kalima, Puhl, and others of his contemporaries. Konjetzny,[3] especially, added a great deal to the knowledge of gastritis, especially as related to peptic ulcer, by his detailed study of about 50 resected stomachs. He was the first to call attention to the fact that practically every case of peptic ulcer showed a chronic antral gastritis, and he stated as his opinion that the gastritis antedated the development of the ulcer.

More recent writers have used much the same criteria for the determination of the presence or absence of gastritis. Notable among these is the work of Hebbel[4] of the University of Minnesota. He studied a control series of 260 stomachs removed at autopsy in individuals free from manifest gastric disease, and 106 stomachs resected for ulcer, and 52 stomachs resected for carcinoma. Whereas, the antral region of the stomach fell in the distinctly abnormal group in only 18.5 per cent of the control group, it did so in the ulcer group in all, or 100 per cent, of the cases. In the body mucosa 22 per cent of the control series fell in group III or the distinctly abnormal group, whereas only 2 of the 78 cases of duodenal ulcer fell in this group. However, there were 14 cases of hypertrophy. In 13 cases of gastric and duodenal ulcers, 6 were in the distinctly abnormal group, 2 showed hypertrophy, and of the 15 cases of gastric ulcer alone, 12 were in the distinctly abnormal group. Hebbel's findings, therefore, confirm the earlier observations of Konjetzny that antral gastritis occurs in practically all peptic ulcer cases. Furthermore, his findings do not agree with those of Hamperl,

Hillenbrand and others who report a high incidence of gastritis in persons free of ulcer or carcinoma.

Our interest in gastritis has arisen largely from observations made of this condition in the gastroscopic clinic in the out-patient department. I think it is generally agreed that the study of gastritis was given added stimulus following the introduction of the Wolf-Schindler flexible gastroscope in 1932. The names of Schindler, Gutzeit, Henning and Moutier stand out as pioneers in this field of investigation.

Gastroscopists have largely adopted Schindler's classification[2] of chronic gastritis as a working classification. This has the disadvantage of being based entirely on gross appearance - as there is no way in which biopsies can be obtained through the flexible gastroscope. It has the advantage of being based on the appearance of living tissue. Schindler claims for it that it corresponds excellently with the histologic classification of Faber. However, he states that it is based almost entirely on the correlation of gastroscopic observations with clinical data.

According to this classification, chronic gastritis is divided into four types: 1) superficial gastritis; 2. atrophic gastritis; 3. hypertrophic gastritis, and 4) gastritis of the postoperative stomach. The fourth or last type is a special form of the disease and will not be discussed here.

In superficial gastritis there is increased redness or hyperemia of the mucosa with edema and the formation of an exudate which can be seen between the folds or covering larger areas of the mucosa. Superficial erosions and hemorrhagic spots may or may not be present. The inflamed mucosa has a tendency to bleed easily from the trauma of the stomach tube or gastroscope.

Atrophic gastritis shows a very characteristic appearance, the most conspicuous feature of which is the thin, flat mucosa which may appear gray or greenish gray in color. Some have referred to this appearance as colorless, rather than trying to describe a definite color. These changes usually appear in patches, but may be diffuse. The atrophic areas may appear depressed. They may be surrounded by normal mucosa or may be combined with areas of superficial gastritis. As the atrophic process progresses, branching, submucosal blood vessels become visible. Schindler refers to this as an infallible sign of atrophy. The atrophic picture becomes more marked, as a rule, in the upper portions of the stomach. Mucosal hemorrhages may occur as well as erosions, but these are not common.

In hypertrophic gastritis, the mucosa first becomes dull and velvety. The process usually starts in the valleys. Later, the folds are thickened, the highlights diminished. As the process increases, the folds become thicker and stiffer and show ridges and creases. The entire mucosa may appear nodular or wartlike. The individual nodes may become large enough to be mistaken for real polyps. It becomes increasingly difficult to flatten the folds by air pressure. There may be erosions or even superficial ulcers. Hemorrhagic spots may be seen.

It is the purpose of this discussion to attempt to correlate the gastroscopic appearance with the histology of the various types of gastritis insofar as this is possible, to bring out attempts which have already been made in this direction, to indicate the problems and difficulties involved and to add some of our own experiences.

As stated above, it must be remembered that gastroscopic diagnosis is based entirely on the gross appearance of the surface of the mucosa. True, one may be aided by other factors such as peristaltic activity, pliability of the stomach wall, etc. The taking of biopsies is not possible and were it possible probably would not be advisable.

It must also be remembered that all parts are not equally well seen through the gastroscope. There are blind spots in all stomachs, large in some, small and quite insignificant in others.

Some parts of the stomach are close to the objective lens of the gastroscope, others quite far away. In some portions the lighting is brilliant, in others we are troubled greatly by shadows and poor lighting. In this connection, it is fundamental to keep in mind that gastroscopic visualization of the antrum of the stomach is far less satisfactory than is that of the corpus. While ulcers and new growths can usually be seen and fairly well described in the antrum, any detailed inspection of the mucosa for changes such as those seen in the various forms of gastritis is more or less impossible.

This leads to consideration of one type of gastritis, and here we discover a negative correlation. I refer to the so-called antral gastritis which has now been rather uniformly accepted by most pathologists as an entity especially in association with peptic ulcer. Most gastroscopists state that they see this condition only infrequently. Schindler[6] and others have tried to explain the discrepancy by ascribing many of the changes seen in surgically resected and autopsy specimens to trauma and post-mortem changes. The very chronicity of the lesions described, as well as their penetration into the depths of the mucosa, make this contention untenable. To me, it is obvious that we shall have to accept the fact, that with the usual gastroscope, at least, the antral mucosa is not seen clearly enough to allow us to recognize the presence of gastritic changes.

It appears, therefore, that when we discuss gastritis from the point of view of its gastroscopic appearance, we are dealing largely with gastritis of the corpus. What studies have been made to attempt to correlate the gastroscopic picture with the histopathology? While the earlier writers on gastroscopy, including Schindler, picture beautifully the histopathology in individual cases, they do not report on any series of cases and give us their over-all experience in such series. Even in a recent article in which his study is based on 15 cases in which biopsies were taken at abdominal operation, 32 cases obtained from gastric resection, and 9 autopsy specimens, Schindler[7] pictures the appearance of individual specimens and then simply makes the statement that a characteristic picture is almost invariably found on microscopic study. In this article, Schindler describes in detail certain cellular changes of the surface epithelium especially in superficial gastritis. As far as I have been able to ascertain, these changes have not been confirmed by pathologists. He suggests also that the transparency of the mucous membrane in atrophic gastritis may be due to "the disappearance of the opaque chief cells". In his description of the histopathology of specimens in which hypertrophic gastritis was diagnosed gastroscopically, he divides these into three types: (1) chronic hypertrophic interstitial gastritis; (2) chronic, hypertrophic, proliferatte gastritis; and (3) chronic, hypertrophic, glandular gastritis.

As far as I know, only 2 reports have appeared in the literature in which series of cases examined gastroscopically have been subject to microscopic study and the over-all correlation between gastroscopic appearance and histopathology given us.

In 1941, Swalm and Morrison[8] examined 25 cases with the flexible gastroscope and followed this by taking punch biopsies through an open end rigid gastroscope. Their studies resulted in agreement between the gastroscopic and histologic diagnosis in 13 cases (52%), Complete disagreement in 6 cases (24%), and questionable correlation in 6 cases (24%). Even though they state that all cases with marked gastroscopic appearance of gastritis were verified histologically, their results must be considered disappointing. Definite histologic corroboration was obtained in two out of three cases of hypertrophic gastritis, six out of eight cases of superficial gastritis, one out of three cases of atrophic gastritis, and three out of eleven normal cases.

In 1943, Benedict and Mallory,[9] studying 51 surgically resected stomachs which had been gastroscoped shortly before surgery, found complete gastroscopic-pathologic agreement in 28 cases (54.9%), partial agreement in 17 cases (33.3%),

or a total of complete or partial agreement in 88.2%. There was complete failure of correlation in 6 cases (11.8%). In breaking down this series into the various types of gastritis, they found agreement in 74.5% of hypertrophic gastritis; 66.7% of superficial gastritis; and 66.7% of atrophic gastritis. While these statistics show some improvement over those of Swalm and Morrison, they still have a good deal to be desired. They claim that the superficial gastritis of the gastroscopist corresponds to the acute exudative gastritis of the pathologist, that the term atrophic gastritis is used the same by both groups, and that hypertrophic gastritis of the gastroscopist corresponds to an exaggerated form of the physiological plasma cell and lymphocytic infiltration of the normal stomach. I believe most authors do not agree with this latter statement and recognize cases in which there is true glandular hyperplasia.

In another type of study, Ruffin and Brown,[10] using controlled air pressures to inflate the stomach in anesthetized dogs as well as human beings, claim that they reproduce the thin appearance of the atrophic mucosa as well as the visible blood vessels simply by raising the air pressure to a certain point. Their studies are not very convincing for two reasons. In the first place, the stomach of the anesthetized dog will allow a much greater air pressure than most humans could tolerate. In the second place, their illustrations never show the complete lack of color so characteristic of atrophic gastritis and the network of blood vessels is extremely faint.

At the gastroscopic clinic in this hospital, studies have been carried out over a period of years which have been previously reported and which indicate certain trends, although they have not attempted to show any gastroscopic-pathologic correlation. In 1940, Carey[11] reported a series of approximately 750 gastroscopic examinations. This showed a total incidence of gastritis without other gastric disease of 44%, including 12.5% atrophic gastritis, 9% superficial gastritis, 22.5% hypertrophic gastritis, These figures agree very well with a similar series studied by Schindler, and reveal the incidence of gastritis as diagnosed gastroscopically in the general gastroscopic population. As contrasted with this, Carey, Wetherby and Ylvisaker,[12] in 1941, reported gastroscopic findings in 233 cases of histamine achlorhydria. In this series, atrophic gastritis was found in 132 cases, superficial gastritis in 44 cases, normal gastric mucosa in 34 cases, carcinoma in 21 cases, and hypertrophic gastritis in 2 cases. Repeat gastric analysis was done in 44 cases. Twenty-one of these showed acid on second test, much to our surprise. Thirteen of these were gastroscoped and only one showed atrophic gastritis and none superficial gastritis. If this relationship held for the entire series, it is easily seen that the incidence of atrophic gastritis and superficial gastritis for the entire series of true achlorhydria would be even higher than that reported above. This high incidence of atrophic and superficial gastritis corresponds with the view of Faber and other pathologists of the association of gastritis and achlorhydria and consitutes indirect evidence of accuracy of the gastroscopic diagnosis.

In 1942, Carey and Ylvisaker[13] reported on the gastroscopic findings in a series of 70 patients with roentgenologically proven duodenal ulcer. Sixteen of these showed a normal gastric mucosa, 27 hypertrophic gastritis, 10 superficial gastritis, 9 gastric ulcer, and 8 condition of hypertonicity and hypersecretion. While this series also shows a high incidence of gastritis, by far the most frequent type is the hypertrophic and there were no cases of atrophic gastritis. This contrasts with the high incidence of atrophic gastritis in the achlorhydria groups. In general, it corresponds with the findings in the corpus in Hebbel's cases. He found 14 cases of hypertrophy in the corpus and only 2 cases of atrophy in his series of 98 duodenal ulcers.

While these indirect evidences of the accuracy of gastroscopic findings in the body of the stomach are significant, gastroscopists are all agreed that

the crying need is for more direct gastroscopic-pathologic correlation. In other words, they realize the importance of determining the histologic appearance for each gastroscopic diagnosis insofar as this is possible.

I will show a few photomicrographs today of cases which have been examined gastroscopically. To show the difficulty of getting sections for study, our records beginning in 1938 and going through 1943 were carefully searched for all those in which a definite diagnosis of atrophic gastritis had been made. Of these cases, all who had come to autopsy or had had gastric resections were looked for. For this entire period only 6 such cases were found in which the pathological material was still available. Two cases of hypertrophic gastritis were added for comparison. All were gastric resection cases. Rolled strips from the entire length of the anterior wall were taken in each case, as this is the part of the stomach best seen through the gastroscope. The strips were cut as far from the new growth or other pathology present as possible.

The first case is that of Mrs. S.S., married, age 6y, who was first seen in the Out-Patient department in March, 1934. Histamine achlorhydria was found. Diagnosis of pernicious anemia was made and she was given parenteral liver extract . She returned in April 1940. Histamine achlorhydria was again present. X-ray examination revealed a carcinoma of the stomach. Gastroscopy on April 10th revealed a nodular growth extending to within 4 cm. of the cardia. Above this there was extensive gastritis with atrophy and a hemorrhagic area with erosion just beyond the cardia. Gastric resection was done April 26, 1940. Section shows severe gastritis throughout with intestinal metaplasia and moderate atrophy. The condition is worst high up in the corpus.

The second case, Mrs. H. J., married, age 60, was first seen in March 1940. Blood normal. Histamine achlorhydria on one occasion. Gastroscopy was done June 5, 1940. This showed a fleshy polyp on the anterior wall just above the angulus. There were slight atrophic changes in the upper part of the stomach. Gastric resection was done June 19, 1940. Section showed Grade III atrophy and grade III gastritis in the corpus. Gastritis and atrophy most pronounced in corpus. Metaplasia most pronounced in antrum.

The third case, Mrs. A. DeB, was first seen June 10, 1940. Blood normal except erythrocytes were down to 3.6. Histamine achlorhydria. X-ray showed a polypoid tumor of the antrum. Gastroscopy on June 19, 1940, showed a polypoid tumor mass in the antrum. In the body of the stomach there was diffuse atrophy, but no other findings. Gastric resection July 2, 1940. Section shows atrophy and gastritis grade III in corpus and intestinal metaplasia most pronounced in antrum.

The fourth case, S. E., white, married, male, age 73, first seen Oct. 3, 1940. Blood normal. Free acid present in gastric contents. Gastroscopy Oct.9th showed a suggestion of a lesion just beyond the angulus which could not be visualized. There was diffuse atrophy throughout the body of the stomach. Gastric resection on Oct. 11th, for benign gastriculcer. Section shows gastritis grade II everywhere. One hemorrhagic erosion. Few spots where atrophy is noticeable, but as a whole not atrophic mucosa.

The fifth case, F.R., white, male, a mild diabetic. Blood normal. Free acid present in gastric contents. Gastroscopy done August 21, 1940, showed an ulcerative lesion with nodular area near the pylorus, and rather extensive atrophy on the anterior wall of the body of the stomach. Gastric resection done, Aug. 29, 1940, for an ulcerating adenocarcinoma. Sections show a grade I gastritis with a grade I - II atrophy in spots, but mostly no atrophy.

The sixth case, G.D., white, married, male, age 55, should not be included here because gastroscopy was done some time after gastric resection for multiple adenocarcinoma. Circulatory changes could have entered the picture and enough time had elapsed for gastritis to have developed. Gastroscopy showed an extreme atrophy of remaining portion of the stomach mucosa. Sections show a severe gastritis with marked atrophy.

In some areas there is so much exudate that the mucosa is not thinned out.

The remaining two cases showed hypertrophic gastritis by gastroscopy and are added just for comparison. On section, the one shows mostly normal mucosa with some areas of hypertrophy but also some areas of atrophy. The other shows almost pure hypertrophic change.

These cases do not constitute a sufficient series to allow us to draw any conclusions. While all the cases diagnosed as showing atrophy on gastroscopy showed some degree of gastritis and atrophy on section, there was a wide range of severity. One case diagnosed slight atrophy showed extensive gastritis and atrophy on section. The others all showed extensive atrophy on gastroscopy, but everything from slight patches to extensive gastritis with atrophy on sections. Probably the only justifiable conclusion from this study is that it indicates a crying need for a great deal more microscopic investigation of gastric mucosae which on gastroscopy show changes, ordinarily classified as atrophic mucosa by this method, in order that we may know what we are looking at.

This presentation should not be closed without a word concerning the relationship of gastritis and gastric cancer, although I doubt that much can be said in the present state of our knowledge. In the past we have read statements by both pathologists and gastroscopists to the effect that carcinoma never develops in a previously normal gastric mucosa, and that it is always preceded by gastric changes. Schindler states that gastric cancer is 3 times as frequent in stomachs showing atrophic gastritis as in those without. The increased incidence of gastric cancer of up to 4 per cent in pernicious anemia in which some degree of gastritis, usually with atrophy, is always present is well known. However, the causal relationship of gastritis and cancer has recently been questioned by several investigators. Guiss and Stewart[14] have recently made an extensive study of the morphologic changes in the gastric mucosa at various ages. They found evidence of atrophic gastritis commonly after the fortieth year; in 66 per cent of cases of death from extragastric cancer; in 82% of apparently normal stomachs, and in 97% of deaths from gastric cancer. They infer that the frequency of chronic gastritis with advancing age may not have any other relationship to gastric cancer than intensification of gastritis by the cancer. Hebbell[4] in 52 stomachs resected for carcinoma, found no evidence to indicate that carcinomas arise with unusual frequency in stomachs already the seat of a diffuse atrophic gastritis. In other words, we still do not know if early carcinoma of the stomach occurs without gastric changes.

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III. GOSSIP

In a book ad in the Journal of the American Medical Association this week the University of Minnesota Hospitals is listed as one of the five foremost medical centers, the other four being Johns Hopkins, Tulane, Washington and Stanford. The ad is about the Medical Clinics number in which Doctors Watson, Hoffbauer, Hall, Flink, Aagaard, Evans, and Spink appear. A few weeks ago reference was made to Dr. Spink in 3 different ads....Edward H. Rynearson of the Mayo Foundation spoke at the Center for Continuation Study this week. He gives a stimulating talk on "Metabolism and Disease". He has a way of using wisecracks to emphasize his points. He honestly believes that the endocrine glands get credit for a lot of things they never do. He doesn't believe the different diagnosis between obesity and myxedema requires a basal metabolism test. He has at last seen 2 women who developed signs of pituitary deficiency following pregnancy. He has been looking for this disease for a long time, as most of the cases he has seen to date have been examples of anorexia nervosa.....Captain Harold S. Adams, Senior Assistant Sanitarian, United States Public Health Service assigned to the Minnesota Department of Health for duty, told us of how he taught food handlers to be more careful when they handled our food dishes, glassware and silverware. He started out in Flint, Michigan where he conducted a one man campaign against dirty habits of food handlers. Some of his pointers included washing hands after going to the toilet, keeping food covered, racking glasses upside down, and many others. He feels rather badly as in spite of everything he has tried to do the University of Minnesota still exposes its glasses to the atmosphere, and incidentally, to the entry of disease agents. Sanitation has been slipping during the war. This story was told. "The boss was in the washroom when one of his employees failed to hand wash before he left. When the boss asked him about it, he said he wasn't going back to work, he was just going out to eat." The outstanding sanitarian in the services is Tom Magath on leave from the Mayo Clinic. When he gets through with an inspection, everyone and everything stays inspected. He has an excellent practical knowledge of bacteriology which he has used to good advantage through the years....There is much talk of protein feeding these days. The University of Minnesota Hospitals series now numbers over 1000 cases. Dick Varco is in constant demand for his teachings in this subject. He does an excellent job, and is much more critical than many others who are applying the method today....Today we started a course in Alcohol and Narcotic Education. We have a state law which compels all teacher training institutions to instruct their students in these subjects. An outline has been written by Doctors Bieter and Potthoff. Dr. Diehl has written the foreword, and Dr. Emerson has added his contribution. Anne Roe is out from Yale to tell us of her evaluation of alcohol education literature. Many writers on this subject spend pages telling of the industrial uses of alcohol in order to appear broad minded. The use of the term "moral" in connection with the problem finds many applications. There is genuine concern today over the large number of individuals who are using drugs to excess. Members of the medical and nursing profession are offenders in this regard. One psychiatrist has reported that 18 physicians and one physician's wife were patients with drug addiction in the last few years. Recent graduates are coming back with the story of addiction. Ease of accessibility and emotional tension, in inadequate individuals are the answers. It is queer how we hesitate to take stands on social problems. Both the alcohol and the drug problem are fairly direct problems. The solution, however, is made complex by our early training and our desire to be well regarded. A mother of a student in my class in Personal Hygiene told me my class was worth the tuition for the quarter because it gave her an idea on how to have a successful family dinner. Other years they had always fought, or someone's feelings were hurt. She tried a little beverage on them and the grouches became happier and the others went to sleep which indicates that there may be a time and place for everything.

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